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THE
LARYNGOSCOPE.

VOL. LIV FEBRUARY, 1944. No. 2

THE CRICOPHARYNGEUS MUSCLE IN ESOPHAGEAL SPEECH.*†

DR. J. R. LINDSAY, DR. RUSSELL H. MORGAN and JOSEPH M. WEPMAN, M.A., Chicago.

The laryngectomized patient is left with four possible means of communication: The use of paper and pencil, the pseudowhisper, the artificial larynx, and the esophageal voice.

Of these four methods the esophageal voice is the most satisfactory both from the psychological and the practical viewpoints since it requires no apparatus, attracting attention to the patient's disability, and is always available. The quality of the voice varies in different subjects, but those who become proficient can carry on intelligible conversation without directing attention to the voice other than that due to its hoarse quality. The ability of nearly all patients to develop a useful pseudovoice after laryngectomy has been demonstrated in many series of cases in the past half-century. The mechanism involved in its production has been the subject of special investigation by numerous authors and was reviewed in 1926 by Hugo Stern.[1] More recently the observations of Thomson,[2] Jackson,[3] Negus,[4,5] Morrison,[6] Stetson,[7] McCall[8] and others have served to provide more exact knowledge of the process.

The origin of the sound which is utilized for producing the pseudovoice has been the subject of considerable debate in former years, but since the observations of Jackson[3] and Negus[4] it has been attributed to the action of the muscular sphincter at the pharyngoesophageal junction known as the cricopharyngeus. Fluoroscopic observations reported by

*Read at the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Cleveland, Jan. 12, 1944.

†From the Division of Otolaryngology and Roentgenology of the University of Chicago.

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Negus,[5] Morrison,[6] McCall[8] and Stetson[7] have demonstrated a narrowing at the region of the pharyngoesophageal junction through which air was expelled from the esophagus to the pharynx during voice production. Stern[1] described the stomach and the esophagus as the reservoirs for air and Morrison[6] drew attention to the momentary dilatation of the pharynx immediately preceding the production of voice and the rapid passage of air into the esophagus as well as into the stomach. McCall[8] noted that during phonation a narrowing could be seen at the pharyngoesophageal junction which in some patients was so local and extreme as to resemble the approximation of true vocal cords.

Recognizing the importance of this sphincter in production of esophageal voice, Negus has recommended special care in preserving the muscle during operation. Although the fluoroscope afforded definite information as to the process of voice production, the various component movements occur so rapidly that a good demonstration and recording by means of Roentgenography has hitherto been lacking.

This method of speech was accurately analyzed by Stetson by recording the pressure changes at the lips, in the mouth and in the trachea and the excursions of the thoracic cage and epigastrium. He was able to demonstrate several important facts, among them that during esophageal speech in the accomplished subjects the interchange of air, as measured at the lips, is quite small. He estimated that only 2.5 to 5 cc. were used for each excursion. He showed also that the part played by the chest and diaphragm in esophageal speech is comparable to that in ordinary speech, except that the respiratory movements are somewhat greater and more irregular during the former.

Following the methods used by Templeton and Kredel[9] for demonstrating the cricopharyngeus muscle by fluoroscopy and Roentgenography, a series of observations were made on laryngectomized patients. The movements of the anatomical parts concerned with esophageal voice were analyzed under the fluoroscope and spot films made to record the various stages of speech.

It is hoped that the Roentgenograms obtained in this way may prove of assistance in explaining the mechanism and in giving early instruction to patients. Also that the method

may afford information relative to the variation in ability to master the technique in different subjects.

The routine method of speech instruction used in these patients includes preoperative preparation of the patient by the instructor. The reason for loss of speech and the mechanism of the new method of speech are thoroughly discussed.

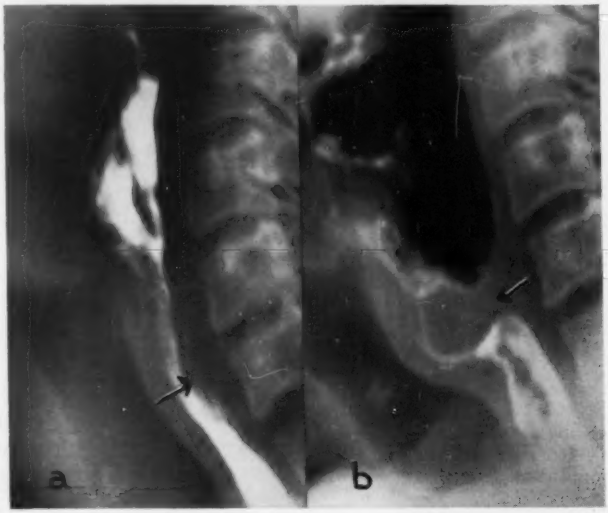

Fig. 1. Pharynx during the resting stage and during the Valsalva test. Lateral projection.

(a) Resting stage during swallowing. A slight anterior curve of the lumen is seen at the pharyngoesophageal junction.

(b) During the Valsalva test the pharynx, upper end of the esophagus and upper end of the trachea are dilated with air and the larynx is displaced forwards. The cricopharyngeus muscle is contracted, forming a closed sphincter with the lumen eccentrically placed against the posterior wall of the larynx.

A previously operated patient with good esophageal speech is introduced and arrangements made for frequent visits by him, as well as the instructor. Experience has taught that in addition to the usual technique of instruction, the psychological effect of the preoperative preparation and the frequent visits by subjects using esophageal speech successfully is of primary importance in attaining success.

Great variation has been noted in the ability of patients to develop the new method of speech. Undoubtedly psychological factors play a large part, but the X-ray observations on

the action of the musculature at the cricopharyngeal junction show considerable variation suggesting a probable causal relationship.

The sphincter is only visible by X-ray in the normal when the larynx is displaced forward away from the postpharyngeal wall. This occurs during introduction of an esophagoscope behind the larynx and prevents passage of the instrument until an effort to swallow causes the sphincter to relax. It also occurs during the performance of the Valsalva experi-

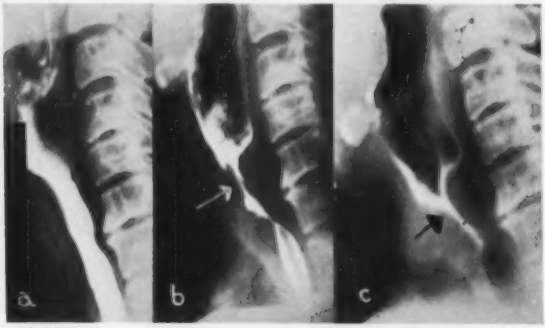

Fig. 2. Pharynx of a laryngectomized patient while learning to swallow and eructate air but before developing speech.

(a) During swallowing. Cricopharyngeus muscle relaxed.

(b) During modified Valsalva test, with tracheotomy open. Air trapped in the pharynx and upper esophagus and pharyngoesophageal sphincter closed.

(c) During Valsalva test with tracheotomy closed. Pharynx and upper esophagus are more dilated and the sphincter more evident. The lumen is less eccentrically placed than before removal of the larynx.

ment with the glottis open (see Fig. 1b). During the swallowing of a large bolus of food the larynx may also be displaced forward but no sign of the cricopharyngeus is evident on X-ray because it is relaxed as the bolus passes down (see Figs. 1a and 2a). Following removal of the larynx and with it the anterior attachments of the muscle, the sphincteric action may be expected to remain inadequate during healing, but as soon as the pharynx is healed the sphincter may again be demonstrated on X-ray.

During the resting stage in the laryngectomized patient the muscle is usually visible as a fairly localized indentation posteriorly (see Fig. 3a). During swallowing the indentation may sometimes persist or it may flatten out as in the normal

(see Figs. 2a and 4b). The appearance may also undergo change during the stages of healing and fibrosis. The sphincteric action of the muscle may be demonstrated by the Valsalva test as soon as the patient has learned to swallow air and force it up again. This involves learning to compress air into the pharynx as the initial part of the swallowing act. By

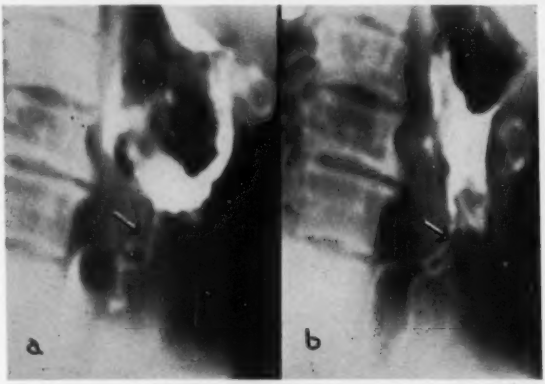

Fig. 3. Pharynx of same patient as Fig. 2 after having learned esophageal speech.

(a) During swallowing or inspiration of air into the esophagus. The pharynx is dilated, the lumen at the sphincter opens (arrow) for a fraction of a second, allowing air to pass down into the upper esophagus.

(b) During phonation. The pharynx is less dilated and the lumen is slightly open at the sphincter (arrow). The cricopharyngeus muscle appears less acutely contracted than in (a) as denoted by the less acute curvature of the lumen.

then keeping air trapped in the pharynx and doing the Valsalva test with the tracheostomy closed by the finger, air in the esophagus is forced upwards against the sphincter, thereby placing it on the stretch whereby it is made visible (see Figs. 2c, 5b, 6b). As the subject masters the act of swallowing and eructating air, the sphincteric action of the cricopharyngeus may be demonstrated by simply having him attempt to "belch" while keeping air trapped in the pharynx (see Fig. 2b). The sphincter is thereby placed on the stretch by the air compressed in the pharynx above and the esophagus below, but to a lesser extent than when intrathoracic pressure is further increased by closure of the tracheostomy (see Figs. 2c, 5b, 6b).

The eructation of air past the partially contracted cricopharyngeus sphincter produces the familar "belching" sound.

To utilize this sound for speech the subject must learn to control it. This seems to be accomplished mainly by acquiring control over the degree of contraction of the cricopharyngeus muscle. During phonation the muscle assumes a somewhat different contour than it does during the Valsalva

Fig. 4. Pharynx of a laryngectomized patient with excellent esophageal voice.

(a) Resting stage.

(b) During swallowing.

test as described above. Instead of a more or less localized narrow sphincter, it may now appear broader in the vertical direction, suggesting a lesser degree of contraction (compare Figs. 5b and 5c).

During phonation it can be seen that the sphincter is not completely closed, leaving a small channel for air to escape.

Although the pitch of the sound produced is very low, it is

not possible to detect vibratory movement of the muscular fold under the fluoroscope; however, with an exposure which varies from one-twentieth to one-thirtieth of a second the Roentgenograms taken during phonation regularly show distinct blurring of the edges of the fold, while those made during resting stage or a modified Valsalva test show clear-

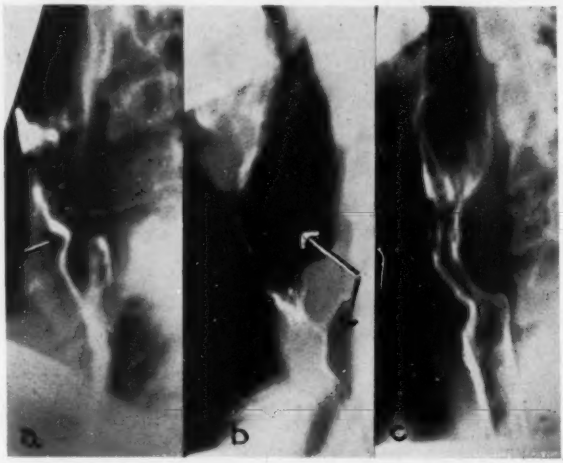

Fig. 5. Pharynx of the same patient as in Fig. 4.

(a) During inspiration or swallowing of air immediately preceding voice production. Movement is so rapid that the outline of the muscle is indistinct. The indentation (arrow) marks the lower border of the sphincter which has opened widely.

(b) During Valsalva test with tracheostomy closed. Dilated pharynx and upper esophagus (arrows).

(c) During voice production. The lumen has blurred edges, due to vibratory movement at the sphincter, and the contour of the muscle is less acutely curved.

cut borders and a completely closed sphincter (compare Figs. 2c and 3 b, also 5b and 5c). It appears clear, therefore, that the movement set up in the borders of the muscular fold by the column of air escaping through the sphincter constitutes the mechanism of sound production.

Observations made on our limited series of subjects seem to indicate that the proficiency of the esophageal voice is related to the degree of control over the cricopharyngeus muscle. The subject with the most efficient voice (see Figs. 4 and 5) showed a greater degree of flexibility of the muscle

during the acts of swallowing. Valsalva test and phonation than was observed in subjects with a lesser degree of proficiency (see Figs. 3 and 6).

To learn esophageal speech the patient must first master the act of swallowing air and causing it to be eructated at will. After having swallowed thick barium paste to outline the walls, the mechanism of air-swallowing may be followed under the fluoroscope. The lips are usually closed at the start but not necessarily so. With the nasopharynx closed off, the tongue goes upwards and backwards, against the palate,

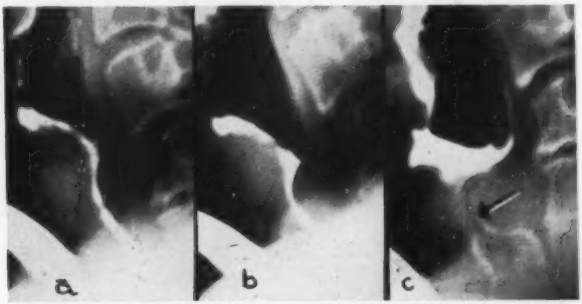

Fig. 6. Pharynx of laryngectomized patient with fair esophageal voice.
(a) Resting stage.
(b) Valsalva test.
(c) During voice production.
The lumen appears slightly open with blurred edges (arrow).

forcing air down into the pharynx, which becomes dilated. The pharyngoesophageal sphincter suddenly opens, allowing air to pass down into the esophagus. The passage of air through the sphincter and upper end of the esophagus occurs in a fraction of a second, but with practice it may be shown on the spot film (see Figs. 3a and 5a). The recording of this process may be facilitated if the radiologist holds a pencil or similar elongated object to the neck of the subject just below the region of the hyoid. During the instant that swallowing occurs, there is a momentary movement of the subject's neck muscles which serves as an excellent signal for making the film. The use of the pencil prevents the radiologist's hand from being exposed to direct X-radiation.

Before swallowing, the esophagus usually contains some air but may be completely collapsed. Immediately after swallowing air and before phonation the esophagus dilates considerably and during phonation the air is forced upward through the sphincter into the larynx (see Figs. 7 and 8). None of the patients examined exhibited any interchange of air at the cardiac sphincter during speech, the esophagus constituting the reservoir for air in all cases.

As the subject becomes skilled, the act of swallowing air becomes more efficient and simplified and is done unconsciously. The pharynx dilates with air, the sphincter opens with flash-like suddenness, allowing air to pass into the upper end of the esophagus, accompanied in the less proficient by a slight sound. The air can then be immediately utilized for production of voice.

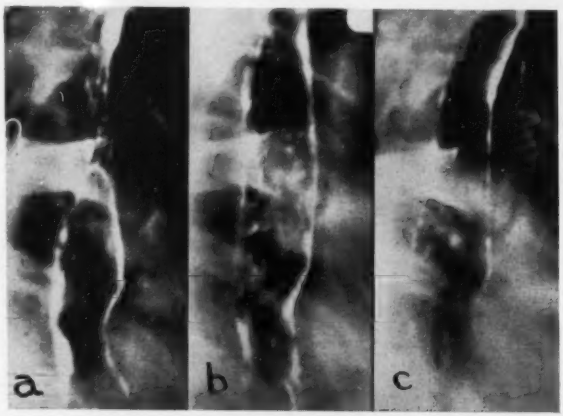

Fig. 7. The esophagus during esophageal voice production.

(a) Resting stage. Moderate amount of air present.

(b) Immediately after swallowing air, the lumen is greatly dilated.

(c) During voice production. The air is forced upwards, and dilatation decreases.

As air passes into the esophagus there is also inspiration of a small amount of air into the trachea. During speech the abdominal muscles contract, the diaphragm rises and air is exhaled from the trachea as well as through the pharynx. In

the accomplished subject the interchange of air both in the trachea and pharynx during speech is quite limited. During phonation, the column of air in the pharynx is narrowed anteroposteriorly but the base of the tongue remains well separated from the posterior pharyngeal wall and does not show evidence of vibratory motion.

SUMMARY.

By means of improved technique it has been possible to analyze with the fluoroscope and record by Roentgenogram the various stages in the mechanism of esophageal voice production.

The function of the pharynx, cricopharyngeus muscle and esophagus in the production of sound has been demonstrated. The Roentgenograms may be of assistance in the early preparation and training of patients for esophageal voice.

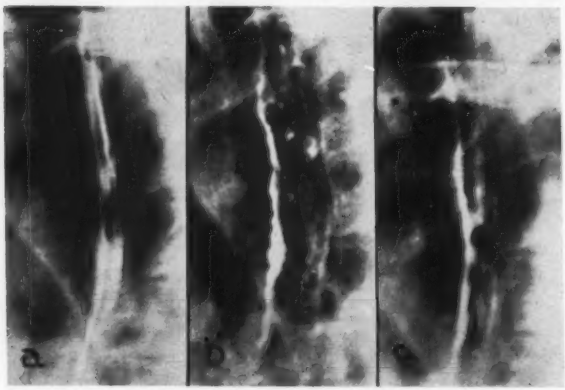

Fig. 8.

(a) Resting stage. Esophagus contracted.

(b) After swallowing air. Marked dilatation.

(c) During voice production. Dilatation decreasing.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be held in New York City at the Waldorf-Astoria Hotel, June 1-2-3-4, 1944.

TORNWALDT'S DISEASE. CHARACTERISTIC HEADACHE SYNDROME AND ETIOLOGY.*

DR. ROBERT W. GODWIN, St. Louis.

The purpose of this paper is to emphasize the association of a characteristic headache syndrome with Tornwaldt's Disease, and to present the pathological findings of excised bursae for their bearing on the etiology of the disease.

LITERATURE.

A brief resumé of the literature reviewed recently by Dorrance,[2] Kully[5] and Woodward[7] is necessary to clarify conflicting concepts of the nature of pharyngeal bursitis.

The term "bursa pharyngea" was first used by Mayer in 1840 in describing a depression found in adults in relationship to the pharyngeal tonsil. The term "bursa" itself is a misnomer, as it is applied to an epithelial evagination rather than to a closed sac lined with synovial membrane which constitutes a true bursa. Mayer did not clearly differentiate this structure from the pouch previously described by Rathke in 1838.

In 1868, von Luschka described an elongated epithelial pocket having an orifice in the midline at the low border of the pharyngeal tonsil and extending backwards to an attachment with the periosteum of the occipital bone. He incorrectly believed this represented a persistence of the oral-hypophysial duct, but gave a complete and accurate description of the pharyngeal bursa.

Pharyngeal cysts in adults have been incorrectly attributed to Seessel's pocket, described in 1877 as arising caudad to Rathke's pouch and to remnants of buccopharyngeal membrane. It has been shown, however, to be a temporary structure pertaining only to the embryo. Arey[1] refers to it as the blind end of entodermal foregut which persists as a pit for a short time after the disappearance of the oral plate.

The term "recessus pharyngeus medius" was first used by Ganghofner in 1879 to refer to the depression in the posterior

*From Department of Otolaryngology, Washington University, and McMillan Hospital, St. Louis.
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part of the median groove of the pharyngeal tonsil. He expressed the belief that the bursa pharyngea was nothing more than a deep median tonsillar fissure bridged over by inflammatory adhesions and enlarged by the retention of secretions.

Tornwaldt, in 1885 and 1887, made clinical observations, by rhinoscopic examination, of openings in the pharyngeal mucosa as described by von Luschka. He noted infection and obstructive cyst formation and associated the condition with headache, frequent upper respiratory infections, asthma and arthritis. This entity has become known clinically as Tornwaldt's Disease.

Studies of a well developed bursa pharyngea in a 38 mm. fetus by Froriep[1] (1882) revealed the intimate relationship between the apex of the bursa and a mass of notochordal tissue. Froriep concluded that notochordal contact had been instrumental in the production of the bursa. The work of Huber,[4] in 1912, confirmed this belief by showing that the pharyngeal bursa is formed at the point where the pharyngeal segment of notochord has retained its union with pharyngeal entoderm. In 1934, Snook[6] studied 201 human embryos, finding pharyngeal bursae present in over 40 per cent of the specimens from 15 mm. to newborn and confirmed the finding of association of the bursa with notochordal tissue.

In 1929, Yankauer[8] reported 155 cases of nasopharyngeal abscesses which he believed were due to retention cysts in the recessus medius of the pharyngeal tonsil rather than to infected pharyngeal bursae. Ninety-three per cent of these patients had not had previous adenoidectomies. Kully,[5] Dorrance[2] and Eagle,[3] however, believed that their clinical findings on many cases suggested infection of pharyngeal bursae rather than adenoid retention cysts or abscesses.

Fig. 1, taken after Arey[1] and Huber,[4] shows the different sites of Rathke's pouch, Seessel's pocket, and the region of the pharynx where notochordal contact with pharyngeal epithelium is thought to produce the pharyngeal bursa.

In Fig. 2, taken after Huber,[4] the pharyngeal bursa is shown in the 32 mm. human embryo with its relationship to the notochordal remnants emerging from the basiocciput. This relationship probably accounts for the attachment of the

apex of the bursa to the periosteum of the occipital bone in adults.

The association of headaches with nasopharyngeal abscesses has been noted by many writers since Tornwaldt's original observations. Of Yankauer's 155 cases, 68 per cent com-

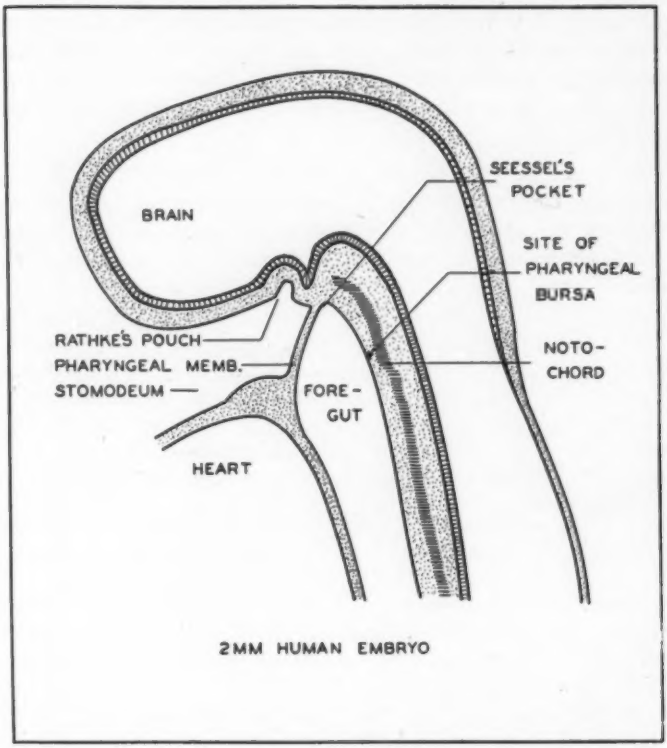

Fig. 1.

plained of headache and neck pain, and about half of these had a characteristic localized occipital headache. Kully[5] found that 14 per cent of his 88 cases complained of this same headache localized below the external occipital protuberance and that many had associated rigidity of the posterior cervical muscles. Eagle[3] reported a case with the same character-

istic headache which was promptly relieved by excising the bursa.

REPORT OF CASES.

The writer's interest in this subject was aroused by the following case:

R. N., a white married woman, 28 years of age, entered Barnes Hospital on neuromedical service for study of a suspected intracranial lesion. She

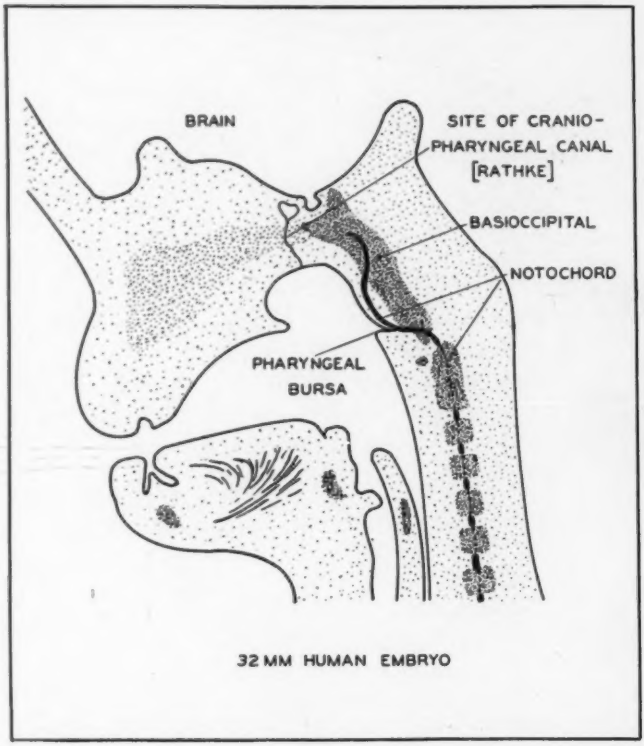

Fig. 2.

gave a history of throbbing and aching suboccipital pain for seven years. The pain localized below the external occipital protuberance and was associated with stiffness and soreness of the posterior cervical muscles. Exacerbations of pain were caused by movement of her head. At frequent intervals she had noted postnasal discharge of thick plugs of mucus difficult to draw back into her throat and at times ran a febrile course of 100-101° F. with exacerbations of the headache. The headaches became

so intense and continuous for two weeks prior to her admission that she was unable to sleep even with sedatives, codeine and aspirin.

There was no associated nausea, vomiting, nasal congestion or colds, scintillations, scotomota, vasomotor changes or allergic symptoms. She had had a tonsillectomy and adenoidectomy performed in childhood.

Complete physical and neurological examination including lumbar puncture, skull films, electrocardiogram, basal metabolic rate, Kahn, N. P. N.,

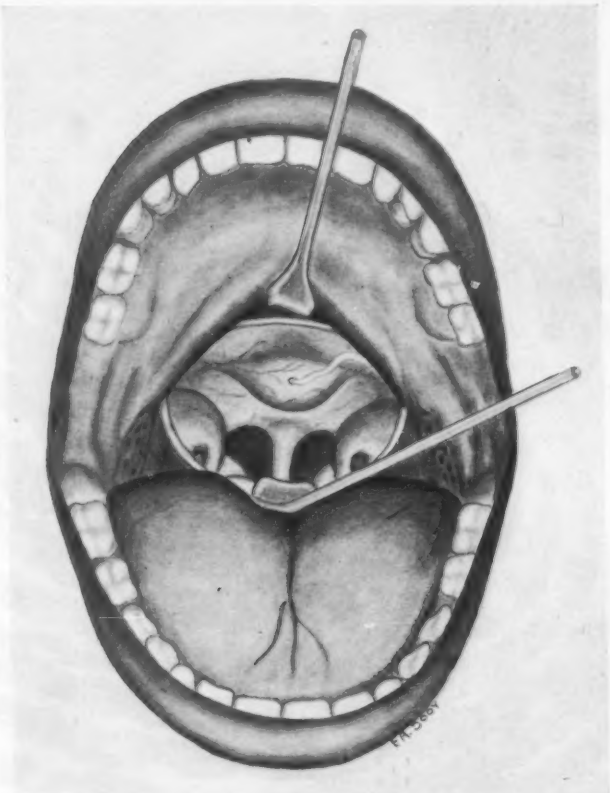

Fig. 3. Semidiagrammatic drawing of cyst formation in pharyngeal bursa as seen on posterior rhinoscopy.

blood sugar, complete blood count and urinalysis revealed essentially normal findings. Her blood pressure was 120/70. Otolaryngological examination revealed a mildly reddened granular pharynx with normal-appearing ears and larynx. On nasopharyngeal mirror examination a swelling, covered by smooth, mildly reddened mucous membrane, was seen in the vault of the nasopharynx. Slightly yellowish, thick mucoid material was exuding from a small midline opening near the lower border of the swelling (see Fig. 3).

Under local anesthesia, with the soft palate retracted, the swelling was digitally expressed with the release of 1 to 2 ccm. of thick yellowish mucoid material. Following this, the patient was free of headaches for the first time in several weeks. The cystic swelling recurred in four days and the top of the cyst was widely excised with a specially sharpened adenoid curette under brief general anesthesia.

The patient has been followed for four months without recurrence of the cyst or the headaches. She has gained weight and has had no further febrile course.

In the past four months seven similar cases were encountered in Washington University Clinic which are believed to represent true pharyngeal bursitis. Five of these cases had smooth mucous membrane-covered swellings as described above. One has a 2 to 3 mm. fistulous opening without a definite swelling, but upon probing, a few drops of yellowish mucoid discharge were released. Another presented a small whitish pustular swelling covered by a brownish crust. In none of these patients was residual adenoid tissue grossly evidenced.

SYMPTOMS.

Five of the seven patients complained of persisting dull aching or throbbing occipital headaches which localized below the external occipital protuberance and were associated with stiffness and soreness of the posterior cervical muscles. Four stated that movement of the head caused exacerbations of the headache.

All of the patients complained of frequent sore throats and postnasal discharge of thick mucus plugs. Two had symptoms associated with Eustachian obstruction, and one had active rheumatoid arthritis. In two patients there was concomitant allergic rhinitis.

ADENOID CYSTS.

During this same period two cases were encountered which conformed to classification as adenoid retention cysts. Both had definite cystic swellings in definite masses of adenoid tissue from which several cubic centimeters of purulent material exuded when the tissue was removed. Both patients had had frequent upper respiratory infections, associated in one

with frontal and interocular headache, but neither had occipital headache nor posterior cervical muscle stiffness.

MICROSCOPIC FINDINGS.

Specimens from surgery bore out the distinction between these two types of cysts. Figs. 4 and 5 show specimens from

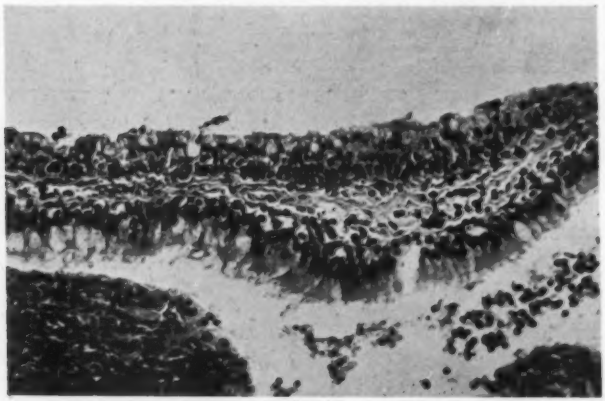

Fig. 4.

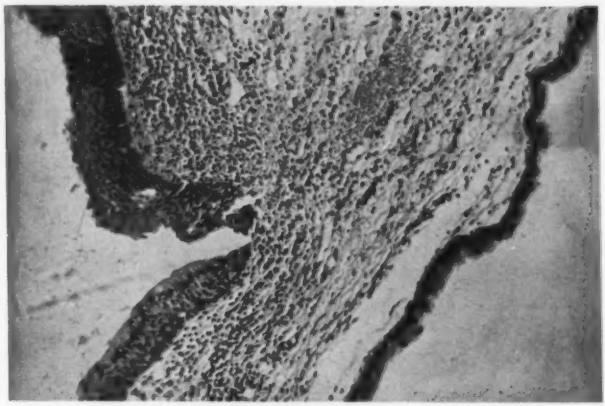

Fig. 5.

Figs. 4 and 5. Photomicrographs of specimens from the surgical excision of pharyngeal bursa cysts.

two of the first seven cases mentioned in which the tops of the smooth swellings were excised with a sharp adenoid

curette. Both sections show pharyngeal mucous membrane on one surface and epithelial cyst lining on the other with intervening connective tissue.

Figs. 6 and 7 show edges of cysts in cases clinically suggesting adenoid cysts. The sections show lymphoid tissue

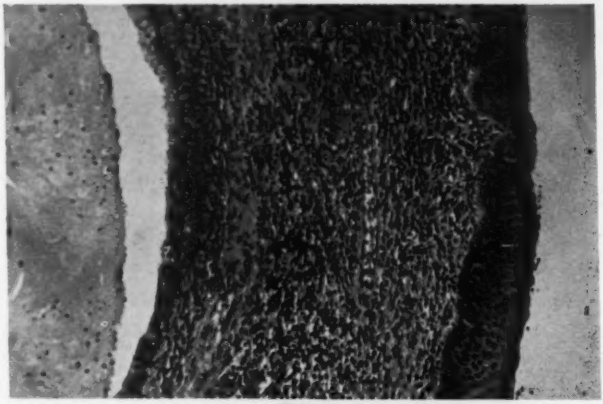

Fig. 6.

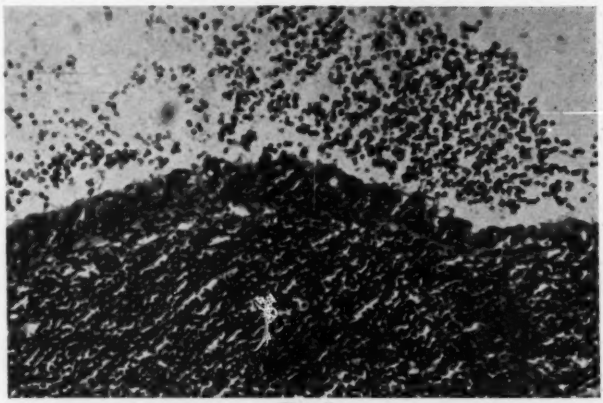

Fig. 7.

Figs. 6 and 7. Photomicrographs of specimens from surgical excision of adenoid retention cysts.

throughout with infected necrotic areas and retention cysts. The adenoid tissue is in immediate approximation with the cystic areas.

SUMMARY AND CONCLUSIONS.

1. Evidence presented indicates that persistent pharyngeal bursae in adults probably arise from notochordal contact with pharyngeal epithelium, rather than arising from Seessel's pocket or Rathke's pouch.

2. Clinical observation corroborated by microscopic sections suggests the existence of cysts in persisting pharyngeal bursae, as well as adenoid retention cysts.

3. A characteristic headache syndrome was observed in five of seven patients in the pharyngeal bursa group. The characteristic symptoms are: *a.* Persisting dull-aching or throbbing occipital headaches localizing below the external occipital protuberance. *b.* Stiffness or soreness of the posterior cervical muscles. *c.* Exacerbations of pain on movement of the head.

These are symptoms that might be expected with an inflammatory process in a pharyngeal bursa with its apex attached to the periosteum of the occipital bone between the attachments of the longus colli muscles. When present the symptoms are so characteristic as to be almost diagnostic of the condition.

4. With special care in nasopharyngeal examination the diagnosis of pharyngeal bursitis may frequently be made. Simple mirror examination is usually adequate, but occasionally soft palate retraction or use of the nasopharyngoscope is necessary.

The writer expresses his appreciation to Dr. Theodore E. Walsh for advice in the preparation of this paper, and to Dr. H. W. Lyman and Dr. William Bryan for the clinical material used in Figs. 5 and 6. Figs. 1, 2 and 3 were drawn by Dr. Francis A. Sooy.

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640 South Kingshighway.

COLLATERAL INFECTION OF THE TEMPORAL BONE IN FULMINATING OSTEOMYELITIS OF THE FRONTAL.*

DR. GEORGE KELEMEN, Boston.

Osteomyelitis of the frontal and osteomyelitis of the temporal bone are, respectively, well defined pathological and clinical entities. Both have been studied to a degree to establish a clear-cut picture for each; then the findings were further examined as to their place amidst other osteomyelitic processes of the surroundings or of more distant parts of the skeleton. Studying these isolated conditions in this way, important data were obtained and a possible connection between the two regional manifestations offered a special interest. Ersner and Winston[2] called attention to some points of importance, as possibility of secondary involvement of the temporal bone, the otic capsule and the auditory nerve by embolic phenomena; by contiguity or toxic absorption in general osteomyelitis. They advise repeated Roentgenological pictures of the temporal bones and otoscopic examinations during any attack of general osteomyelitis.

Forty years ago, Schilling[15] gave a common discussion of the osteomyelitis of the bones of the skull secondary to inflammations of the frontal sinus and the middle ear. Two of his nine cases were related to both regions, the ear process following the frontal manifestations,respectively after eight and five months, both with fatal termination. In one of his cases, the diffuse meningitis was connected with extension from the temporal bone into the middle cranial fossa; in the other with a thrombosis of the lateral sinus.

Definite manifestations in both regions demonstrate clearly their connection, but besides these examples of fully developed complications, additional study seems to suggest itself. This is to start from findings where only one of the two areas in question shows serious changes, while in the other there are only initial, minimal clinical signs. The following history

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may help to trace the pathway of the process from an osteomyelitic frontal in the direction of the temporal bone.

A white boy (No. 363290), age 14 years, was admitted with the past history of having the usual diseases of childhood without sequelae. He had allergic rhinitis, seasonal in type, beginning in August; right otitis media in infancy but without any complications.

Present Illness: On June 18, 1942, he went swimming, wearing a well fitted nose clip. Three days later he complained of marked nasal obstruction. This was followed the next day by pain over the right eye, with headache and fever. As the day went on, the pain and temperature increased. On the fifth day he was seen by the family doctor who made the diagnosis of streptococcus pharyngitis. On the seventh day there was marked swelling with intense pain over the entire forehead.

He was admitted to the local hospital, where a transverse incision 5 inches long was made across the brow. Much pus was evacuated and a perforation was noted in the bone of the right frontal sinus. Considerable pus welled up from this perforation. He felt much better for several days after drainage was established. The temperature continued between 100° and 103.5.° The edema had extended to the right side of his face and upward over the scalp. He was seen by Dr. Hahn, a neurosurgeon, who made a diagnosis of osteomyelitis of the skull with a possible extradural abscess. There was no evidence of intracranial extension at this examination. He was referred to the Massachusetts Eye and Ear Infirmary to the care of Dr. P. E. Meltzer.

He was admitted July 1, 1942, and examination revealed a well developed boy, alert, intelligent and co-operative. Skin warm and dry, temperature 102; pulse 116; respiration 22. He had purulent nasal discharge with marked swelling of all the turbinates. There was edema of both upper lids and forehead, extending upward over the hair line, tender and painful to touch. There was edema also over the right side of the face, zygomatic region particularly with trismus. Heart, lungs negative. Blood pressure 115/60. R. B. C. 3,460,000. W. B. C. 22,100.

X-ray Report: Osteomyelitis of the front wall of both frontals with extension out on the right side into the temporoparietal region. Sinuses all filled with pus.

He was operated upon on that day.

Operation: Ether anesthesia. Operator: Dr. Meltzer. A pre-existing incision about 5 inches long, one-half inch over both eyebrows, was found, from which a thick purulent discharge exuded. The transverse incision was extended laterally well into the temporal muscle on each side. A sagittal incision at right angles to the transverse incision was made in a posterior direction. The flaps were laid back, exposing a perforation through the anterior wall of the frontal on the right side. An area of necrotic bone at the external angular process of the right frontal with multiple perforations was seen. At this area a lead was found into a large subtemporal abscess, which extended to the coronoid process of the mandible. Examination of the mucous membrane of the frontal sinus showed marked edema; removal of the posterior wall of the frontal revealed no extradural abscess. By following a lead of a partly thrombosed dural vessel a large extradural abscess was uncovered beyond the confines of the frontal sinus. The frontal bone was then entirely removed, as was a large portion of the parietal. At no time in the removal of the bone was it felt that one reached beyond infected diploe, because when the bone was removed, it would appear macroscopically healthy, but on waiting several moments, tiny droplets of pus would appear from the marrow spaces. The operation was completed and 6 gm. of sulfanilamide

powder spread around the defect, over the dura and into the subtemporal abscess.

The boy appeared better but the temperature remained at 103° continuously. The pulse ranged from 90 to 110. Dressings were done daily in which 2 gm. of sulfanilamide were spread throughout the wound. After the first day practically no suppuration was noted about the wound.

July 5: Fundi O. D. normal. O. S. Disc appears slightly blurred above and below and there is definite temporal pallor (Dr. Liebmann). Lumbar puncture: 122 cc. clear fluid. Initial pressure: 240; final pressure: 160.

On July 6, because of signs indicating spread of the process intradurally, he was seen by a neurologist, Dr. Kubik. The essentials of his report follow: Increasing headaches, most severe in right posterior parietal and occipital region. Drowsiness the past 24 hours.

Findings: Marked rigidity of the neck. Orbital swelling at the right. Fairly rapid convulsive twitching of the facial muscles to the left side of the nose. Slight left facial weakness. Left arm tends to fall away very slightly several times. Tendon reflex a shade more active at the left. Impression: History and findings very suggestive of subdural abscess. Transplants from blood culture: anaerobic: staphylococcus albus; aerobic: staphylococcus aureus. Through constant medication with sulfadiazine an average level of about 16 mgm. was maintained. X-ray showed further extension of the osteomyelitic process into the occipital bone; locally, however, no evidence of pus.

July 6: Exploration for subdural abscess: Because the extradural abscess found at the first operation was near the midline of the right frontal lobe, an incision was made through the dura of that area. A quantity of pus escaped, perhaps two teaspoonfuls. The extent of the subdural abscess could be outlined by passing a flat spatula through the incision into a well encapsulated cavity. A counter-incision was then made into the most dependent portion of the abscess and a through and through drain inserted.

Signs and symptoms of diffuse meningitis became evident July 9; death on July 11.

The essentials of the findings at autopsy, by Dr. Tracy B. Mallory, were as follows: Operative defect is covered with gray granulation tissue. Tract of the rubber drain tube is covered with gray-green granulation tissue. The skull bones that remain are normal in appearance. There is a small amount of pus between the skull bones and the dura mater. The superior sagittal sinus contains a clot which has the appearance of antemortem formation. There is milky thickening over the superior right hemisphere and the superior part of the left frontal lobe. When the dura mater is incised there is an escape of a considerable amount of gray-green pus from the subdural space. There is a milky thickening of arachnoid over both hemispheres, particularly prominent in the interpeduncular spaces. The ethmoid and sphenoid sinuses contain considerable quantity of gray-brown, tenacious, mucopurulent material. The mastoid sinuses contain a slight amount of brown, tenacious, mucopurulent material.

Brain (R. D. Adams): The outer surface of the arachnoid over both frontal lobes is covered with a yellowish green material which can be removed easily, leaving a fairly clean surface. On the right side this material is present not only on the lateral surface of the frontal lobe, but also on the orbital and mesial surfaces. In the pole of the right frontal lobe there is a triangular-shaped softening measuring 25 x 3 cm. in the horizontal plane, within which are at least two small abscesses, the largest being 8 cm. in diameter. There are hemorrhages in the cortex of the involved area and hemorrhagic discoloration of the white matter. The entire ventricular system is displaced to the left. Microscopically: There is an extensive subdural exudate composed predominantly of poly-

morphonuclears. Large clumps of cocci are seen throughout the exudate. There is no organization of the exudate immediately beneath the dura, but there is definite fibroblastic proliferation of the pia arachnoid. There is a large area of necrotic tissue in which there are two rather poorly encapsulated abscesses, one of which comes to within a few millimeters of the pia. Many of the cerebral veins are occluded by masses of cocci and surrounded by a heavy mantle of polymorphonuclears or an abscess. The nerve and glia cells are undergoing ischemic necrosis. Several small hemorrhages are scattered through the cortex and subcortical white matter, especially near the abscesses. There is a leptomeningitis of polymorphonuclears, lymphocytes and monocytes over the medulla.

Roentgenological examination of the calvarium postmortem revealed no abnormality, as moth-eaten appearance, etc.

The temporal bones were removed and after decalcification cut into a series of horizontal sections.

Right Temporal Bone: The external canal wall, including its lining, is normal, as is the tympanic membrane. The tegmen antri and tegmen tympani are thoroughly pneumatized. Some of the pneumatic cells, but not all, contain transudate; others contain exudate of a fibrinous-foamy appearance. In some cells there is but a minimal infiltration with polymorphonuclears. In other cells one sees a beginning resorption originating from the mucosal lining of the cell wall. This mucous membrane is hyperemic and so is the lining of the tympanic cavity. The fibrinous exudate which covers the promontorial wall and some of the ligaments of the ossicles forms larger accumulations against the wall of the attic and on the surface of the secondary tympanic membrane. In many of the pneumatic cells of the tegmen there is a thickening of the mucosa caused by proliferation of connective tissue, considerably narrowing down the lumen of the cell. The same condition is found sporadically in the perilabyrinthine cells but is not present in the main tympanic cavity. Larger residua, pointing to previous inflammatory processes, are absent. In the tegmen, at about the boundary of tegmen antri and tegmen tympani, an intercellular wall presents a peculiar transformation into reticulized bone with intensive apposition and resorption. (A detailed description of this part is given elsewhere.[7]) The petrous tip consists of cancellous bone, the marrow spaces showing an entirely normal picture. Normal conditions prevail in the marrow spaces around the bulb, the carotid and the facial canal. There are thrombi in the veins of the pericarotic plexus. The labyrinthine end-organs are poorly conserved but show no pathological changes. Between the wall of the internal acoustic meatus and the VIIIth nerve there is some accumulation of exudate.

Left Temporal Bone: As in the right temporal bone, the pathological findings are essentially limited to the cells of the tegmen, which, however, is pneumatized to a lesser degree than the tegmen on the right side. There are more cells which contain a transudate or an exudate, the latter poor as to cellular content. Within the exudate are spaces such as might be left empty by cholesterol crystals. This condition progresses further towards the inner ear; an exudate with generally scarce cell infiltration is found in the perilabyrinthine pneumatic cells and can be followed downward to the level of the hypotympanum. In some pneumatic spaces the exudate contains small free hemorrhages. The thickening of the mucous membrane caused by proliferating connective tissue in the cells is much less evident than on the right side and is entirely restricted to the pneumatic spaces of the tegmen.

COMMENT.

Discussing the possible connections between osteomyelitis in the frontal and in the temporal bone, several explanations

help us to gain a clear-cut picture. Mellinger[11] stated that of all the bones of the skull, the temporal is most frequently attacked by an osteomyelitic process. In his opinion, the limitation to an individual bone is caused by sutures which tend

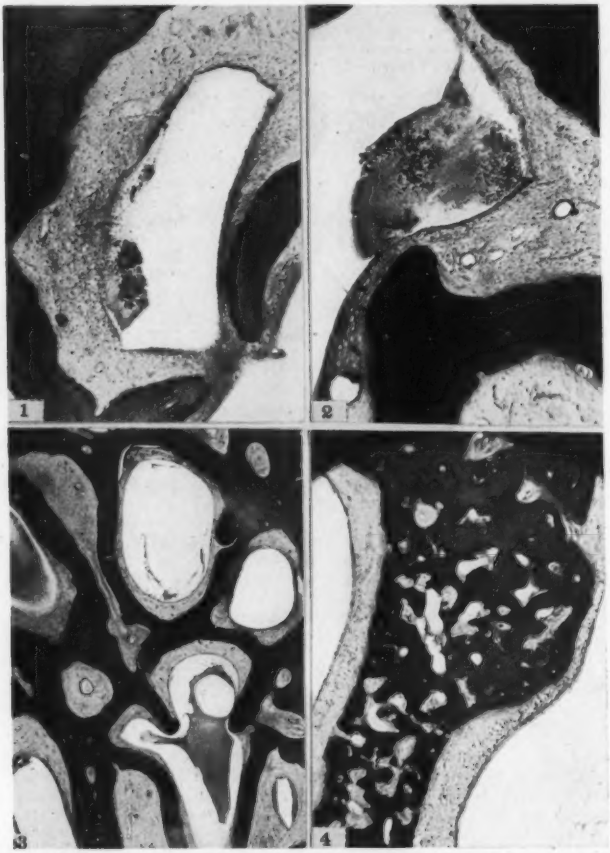

Fig. 1. Right temporal bone. Exudate in cell of tegmen tympani, beginning of resorption. Mucous membrane of pneumatic cell thickened with round cell infiltration.

Fig. 2. Right temporal bone. Exudate in cell of tegmen tympani, beginning of resorption. Mucous membrane of pneumatic cell thickened.

Fig. 3. Left temporal bone. Perilabyrinthine pneumatic cells in the level of the hypotympanum. Some cells contain transudate; some others exudate. Slight hyperemia of the mucous membrane of the cells.

Fig. 4. Right temporal bone. Peculiar reticulization with tumor-like swelling in one of the intercellular walls of the tegmen.

to remain open throughout life. This concept emphasizes the anatomical conditions as directing the course of the process. As a consequence of Mellinger's considerations, we must assume that the limiting rôle of the sutures works both ways in blocking an extension. Furstenberg[4] found clinical and pathological evidence that at least for a time the sutures occasionally tend to arrest the process of the infection. Mosher and Judd[14] present a picture (see Fig. 15 of their publication) which shows that a suture in the adult is only a partial barrier. In their opinion, the suture lines may act as a barrier to the progressive infection but it is certainly not very effective.

In the concept of others, anatomical limitations are disregarded. Ersner and Winston[2] claim that in osteomyelitis other than that of the temporal bone the involvement occurs by embolic phenomena or toxic absorption. Brunner[1] analyzed a case, including microscopical findings, where osteomyelitis of the frontal bone of sinus origin was followed by direct extension of the process into the right ear, while the infection was carried into the left ear by the blood stream. He stated that in osteomyelitis of the skull the temporal bone may become infected either by continuity or by a blood stream infection.

"Primary" and "secondary" in osteomyelitis of the temporal bone were given different definitions. Mayer[10] called an osteomyelitis secondary when its origin is outside the temporal bone; but after involving the latter it respects no limits and extends even beyond the boundaries of the temporal bone on some other side. Kelemen[5,6] called the osteomyelitis of the temporal bone secondary insofar as it follows a pneumatic-otitic process, with later encroachment of the surrounding bones, while the original otitic bone process remains necessarily confined to the pneumatized region.

Lack of continuity is characteristic for both processes, the pneumatic-otitic and the osteomyelitic. According to Brunner,[1] the metastatic otitis media in these cases does not involve the entire mucous membrane of tube, middle ear and antrum; it has, he claims, a "spot-like" appearance. This was verified by the findings in both temporal bones in our case. Osteomyelitic changes were demonstrated by Kelemen[5] at different parts of the cranial base without apparent direct connection

with the main focus. The explanation is, for both cases, propagation by the blood stream. This is the main vehicle for the spread of infection in osteomyelitis of the frontal. The chief pathway, as stated by Mosher,[13] is by way of the diploic veins and the spread of the infection is due to the number and size of the veins.

The findings as offered by the present case yield the following information as to reconstruction of a pathway between the frontal and temporal bones. At the operation an area of necrotic bone was found at the outer angle of the frontal (zygomatic process). Together with the frontal bone, a large part of the parietal had to be removed, as the pathological changes could be followed into this bone. The coronal suture proved powerless to limit the spread and by the same token the squamosal suture was no more effective in protecting the temporal. The latter was involved, too, but the pathological manifestations were confined to the pneumatic system; the autopsy showed pus in the mastoid and the microscopical examination revealed pathology in the pneumatic spaces of the middle ear. Here again, the petrosquamosal suture was ineffective, as were the coronal and the squamosal. In this region the pathway was uninterrupted beginning in the paranasal sinuses and ending in the perilabyrinthine cells of both temporals.

These topographical considerations have to be followed as to arrangement and pneumatization of the cells. Arrangement, as demonstrated by Meltzer,[12] depends on the position of the lateral sinus, while the type of pneumatization and character of the epithelium is influenced by inheritance and postnatal inflammations. The temporal bones examined here yielded a picture of excellent pneumatization, only slightly less on the left side. This barrier of pneumatized bone blocked the osteomyelitic process in its progress from the cancellous parietal into the cancellous part of the temporal around the petrous tip.

Mosher[13] stated that he has learned to be suspicious of the outer angle of the frontal sinus and of the external angle of the frontal bone. The possibility of encroachment on the anterior limit of the temporal fossa is obvious, especially when one remembers the statement of Mosher[13] that infec-

tion of the medulla of the bone extends 1 to 1½ inches beyond the area of necrosis. Thus the borderline of the pneumatized area is easily reached by the osteomyelitic process in its descent from the parietal.

At the age of our patient — 14 years — the intimate connection between mucosa of the pneumatic cells and marrow of the cancellous bone had already ceased a long time ago. This is an important factor in early childhood which facilitates propagation, not only in the direction of the petrous tip but also into the cancellous bone of the temporal squama; both complications are equally severe.

The character of the pathological changes in the temporal bone itself was mucopurulent material in the mastoid cells as revealed by the autopsy, and a transudate and exudate, partly hemorrhagic, in the air spaces of the middle ear, besides hyperemia of the mucosal lining of the latter. In some of the cells beginning resorption was seen. From the wall of the pneumatic cell, originating in the submucosa, vasculated projections reached into the mass of the exudate. The latter contained polymorphonuclears, lymphocytes, some of them considerably swollen; also noted were "caudate" cells, derivatives of the epithelium, all imbedded in fibrin. These are recent phenomena, collateral to the main inflammation in the calvarium. The production of transudate, transformation into exudate by immigrating cells and simultaneous resorption, are proceeding along a parallel much in the same manner as the osteomyelitic changes include simultaneous sequestration and osteoblastic apposition (Kelemen,[6] Fig. 9).

But there were other important findings in the pneumatic cells. Signs of organization were very evident on the right side at the level of the tegmen, absent in the tympanic cavity, sporadic in the perilabyrinthine cells. On the left side they were infrequent even in the cells of the tegmen. These cushions of dense connective tissue frequently narrow down considerably the lumen of the individual pneumatic cell, and gain importance in view of the findings of Mosher[13] and Kos,[8] who, in osteomyelitis of the frontal found early demarcation by newly formed solid connective tissue barriers. This condition of the mucous membrane: considerable thickening by proliferation of connective tissue with signs of resorption of

the contents in the lumen, offers the histological picture of a subacute otitis media in the initial stage of organization. The accumulations of connective tissue are not residua of previous processes as they are not found in the predilectional locations, as are the niches and periligamentous spaces of the tympanic cavity.

In this way formation of connective tissue barriers is a common aspect of the pneumatic-otitic process in the temporal bone *and* the osteomyelitic process in the frontal bone; but in the further course of the healing process this tissue will for its greater part disappear from the pneumatic cell by resorption, whereas in the frontal it will become an important factor promoting recalcification (healing by recalcification, as demonstrated in Case 2 of Fred's[3] report on osteomyelitis of the frontal bone).

The defense apparatus of the mucosa in the pneumatic cells was in perfect shape to block the progression by its reactive measures. That this defense can be a very effective one has often been demonstrated. MacMillan[9] reported his Roentgenological experiences regarding the pneumatic mastoid in the following way: "It is remarkable that spreading osteomyelitis is encountered so infrequently in this area. I have observed this condition only three times in approximately thirty thousand examinations."

It was shown that the meninges are more liable to become infected from an osteomyelitic focus than from suppurating pneumatic cells. In the latter the propagation of the infection is slower or perhaps there are more vascular channels which lead into the subdural space from cancellous bone. Instead of more attempts at direct extension as manifested by perforations of "fruste" form, characterizing the pneumatic-otitic process, in osteomyelitis the cortex is carried away in large sequesters widely opening the pathway into the endocranium. In the present case, the meningitis found at the autopsy extended to the right side over the surface of the temporal and parietal lobes. The epidural and subdural abscesses with their collateral changes in the brain were secondary to the osteomyelitic process in the frontal bone and did not play any part in the extension through the bones of the calvarium. The pus found in the internal auditory meatus

originated from the meningitis and not from an extension through the cortex of the temporal bone.

SUMMARY.

Microscopical examination of the temporal bones of a 14-year-old boy with osteomyelitis of the frontal and parietal bones of frontal sinus origin revealed a hemorrhagic exudate with thickening of the mucosa produced by proliferating connective tissue in the pneumatic spaces of the middle ear on both sides. It was possible to reconstruct a pathway beginning in the paranasal sinuses, across the frontal, parietal and temporal bones. Reaching the pneumatized area of the temporal, the osteomyelitic process gave way to a pneumatic-otitic one. The latter involved the entire pneumatic system from the mastoidal to the perilabyrinthine cells and could be classified pathohistologically as a subacute exudative otitis media.

The comparatively infrequent mutual involvement of the temporal and frontal bones in osteomyelitic processes is explained by blockage of the respective areas by the interlying penumatic system of the middle ear. A collateral otitic process develops in the pneumatic cells with mobilization of the defense mechanism, tending to limit the spread of the osteomyelitis. When this is accomplished, the inflammation stops at the boundary of the pneumatic system.

Formation of abundant connective tissue is evidently the initial phase of healing in both conditions; but, whereas this promotes recalcification in frontal osteomyelitis, the surplus connective tissue is resorbed later in the course of the reconstructive phase in the pneumatic-otitic process.

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414 Beacon Street.

TREATMENT OF LARYNGOTRACHEOBRONCHITIS.*

DR. THEO. E. WALSH, St. Louis.

Because laryngotracheobronchitis is so serious a disease, its further consideration is justifiable in spite of the many excellent articles of recent years. It is the object of this communication to emphasize a variety of the disease in which the infection seems rather to ascend from bronchioles to trachea and larynx; in which laryngeal obstruction is not the paramount cause of the dyspnea and in which early tracheotomy has proven of value, not for the relief of laryngeal obstruction but for the removal, by aspiration, of tracheal and bronchial secretions.

SYMPTOMATOLOGY.

Vivid and careful description of the symptoms of this disease have been written by Gittins, Richards, Orton and others. They have described the process as starting in the upper respiratory tract, involving the larynx and trachea and later the bronchi. In most cases laryngeal obstruction and the presence of crusts and thick mucoid secretions in the trachea were prominent symptoms.

In this series of cases the disease started as a relatively mild "cold." This was followed in about a week by increasing dyspnea, the patients became toxic and, as the dyspnea continued, more and more exhausted.

In those cases which were seen early, the findings were those of bronchiolitis; there was moderate dehydration; moderate dyspnea and toxicity; the temperature was between 38° and 39° C.

Examination revealed moderate inflammation of the upper respiratory passages, and breath sounds in both lungs were diminished and of a wheezing character. Respirations and pulse rate were rapid.

In the majority of cases, however, the patients were seen after they had experienced increasing dyspnea for two or three days or longer. Toxicity and dehydration were marked;

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temperature was between 39° and 40° C.; retraction of the intercostal spaces, suprasternal notch and the xiphoid was marked; the children appeared exhausted, restless and anxious. The breath sounds were absent in some areas and in others sticky rales and coarse ronchi and wheezes were heard. Laryngeal edema was present but was not the cause of the marked dyspnea. Cyanosis was present in some cases; the pallor which is seen so often with exhaustion due to respiratory obstruction was more frequent and was considered a danger sign.

PATHOLOGY.

Richards[1] described the pathologic findings in this condition in an admirable paper. Orton[2] reports eight autopsies in laryngotracheobronchitis and confirms Richard's descriptions. The findings in the fatal cases in this series agree with theirs.

Emphasis should be placed on: *a.* The destruction of the cilia and epithelium of the tracheobronchial tree; *b.* the presence of tenacious thick mucus and pus, which in severe cases seemed to be fixed to the goblet cells of the remaining epithelium, as Hilding[3] has described; *c.* the marked edema of the tissues of the trachea and bronchi (see Figs. 1, 2, 3 and 4).

ETIOLOGY.

The predominating organism cultured from the tracheal secretions was a staphylococcus. It was usually found in combination with other bacteria such as pneumococci and streptococci. Other organisms found were: B. Influenzae (three cases); M. Pharyngis siccus (one case) and Friedlander's Bacillus (one case).

TREATMENT.

The rationale of therapy was based on: *1.* Combating the infection and toxicity; *2.* prevention of exhaustion; *3.* maintenance of adequate airway.

On admission patients were given fluids by hypodermoclysis and full doses of a sulfonamide drug. The initial dose of the drug was given as the sodium salt in the fluids administered parenterally. The initial dose of drug was 0.1 gm. per kilogram of body weight and was followed by the appropriate

dose each four hours so that the total for the first 24 hours was equivalent to 0.3 gm. per kilo, and for the succeeding days, 0.2 gm. per kilo in 24 hours. After the initial dose the

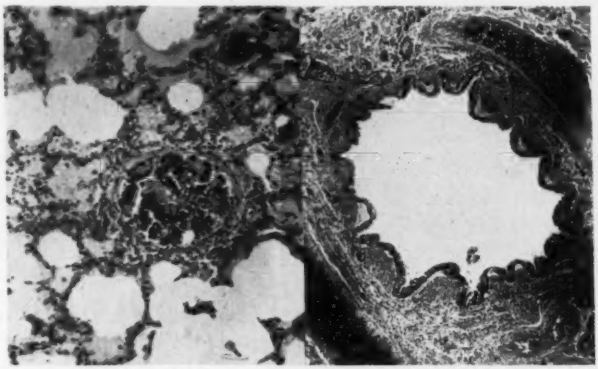

Fig. 1. Fig. 2.

Fig. 1. Microphotograph of a small bronchus. 135x. Note the desquamated epithelium and mucus occluding the lumen.

Fig. 2. Microphotograph of a small bronchus. 40x. Note the edema and the beginning separation of the epithelium.

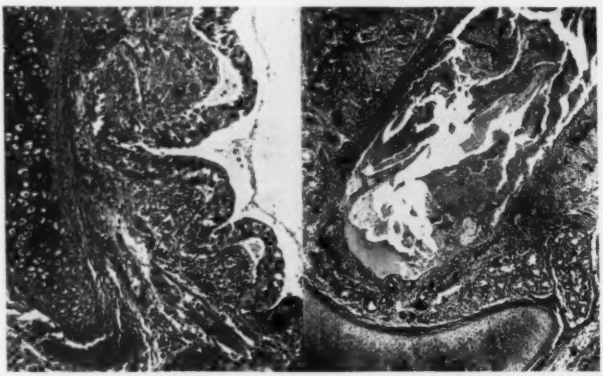

Fig. 3. Fig. 4.

Fig. 3. Microphotograph 135x. High magnification of Fig. 2.

Fig. 4. Microphotograph 35x. Large bronchus filled with exudate and desquamated epithelium.

drug was given by mouth. With the patient adequately hydrated no untoward effects were noticed. Daily urinalysis and blood counts were made and the drug concentration in

the blood determined. Where indicated, blood transfusions were given. The sulfonamide most used in this series was sulfadiazine; in 1940 and 1941, sulfathiazole was used in some cases, and a few patients received sulfapyridine and sulfapyrizine.

Immediately on admission the patients were placed in an adequately cooled and moistened atmosphere. It was found difficult to obtain ideal conditions, with the facilities available, without the use of the Burgess box. This apparatus consists of a tent which is placed over the patient in bed. It has ice in a container in the headpiece and through this ice, oxygen and carbon dioxide may be piped. It was found that pure oxygen was not desirable as it tended to cause more tracheal crusting, as Holinger[4] has pointed out. A carbon dioxide 5 per cent and oxygen 95 per cent mixture was used. The child was left at rest and disturbed as little as possible.

Under these conditions many patients showed marked improvement in from one to two hours. Respirations became less labored; retraction, although still present, was less marked; patients were more at ease and less anxious. In such cases, with continuation of therapy, cure resulted in from four to 10 days.

In many cases (35 per cent), however, improvement was not obtained under ideal conditions of therapy. The respirations and pulse rate increased and retraction was not lessened. In such cases it was felt that prompt tracheotomy was advisable. A bronchoscope was passed and suction of the trachea and bronchi effected. The fact that the passage of the bronchoscope alone did not relieve the respiratory embarrassment in many cases was evidence that laryngeal edema was not the major cause of the obstructed airway.

The tracheotomy was performed over the bronchoscope and as soon as the tube was inserted isotonic saline with neosynephrine or tuamine was dropped into the trachea and suction with catheter was instituted. It was not until thick mucus and crusts had been removed that the child relaxed and respirations became easy.

The postoperative treatment of tracheotomy is most important. All authors have emphasized the various factors such

as adequate temperature and humidity in the room, the attendance of properly trained nurses, the availability of suction and bronchoscope, the care of the tracheotomy tube, etc.; these factors hardly need reiteration here. A few points, however, in the care of tracheotomized patients with laryngotracheobronchitis deserve emphasis: It is necessary to have a trained house staff constantly on call and available at a moment's notice. It may be necessary to do a bronchoscopic aspiration or to remove crusts at any time and this must be immediately possible. The possibility of crusting is dramatically decreased by the frequent use of saline and a vasoconstrictor in the trachea with suction. Rest is essential if the patient is to recover; even the cleansing of the tracheotomy tube can be done without too much disturbance to the patient.

Decannulization was effected in from four to 31 days in this series, the average being 14 days. The usual routine of a diminishing size tube with final corking of the tube was used. All wounds healed without difficulty.

TABLE I.

Year	Total Cases	Tracheotomy	Deaths in Tracheotomy	Gross Mortality
1940	16	5	0	6%
1941	26	7	0	7.6%
1942	59	23	5 (21.7%)	11.8%
Totals 1940—1942	101	35	4 (14.3%)	10%

RESULTS.

The results in this series, following the principles of therapy outlined above, were very satisfactory. They are shown in Table I. The mortality rate of 14 per cent in tracheotomized cases compares favorably with other reported results.

There can be no question that the severity of epidemics of laryngotracheobronchitis varies from year to year. Gittins[5] mentions this point and it must be seriously considered in evaluating the results of therapy. It seems probable, how-

ever, that the improved results in the last few years can to some extent be laid to the proper use of the sulfonamides.

The presence of a trained otolaryngologic house staff is probably another factor in the favorable results. In several instances death was prevented by the prompt bronchoscopic aspiration of secretions and the removal of crusts.

SUMMARY AND CONCLUSIONS.

1. The treatment of tracheolaryngobronchitis resolves itself into:

a. Combating infection with sulfonamide drugs.

b. Supportive therapy with adequate fluids and ideal conditions for rest.

c. Maintenance of adequate airway, this being best accomplished by early tracheotomy and frequent aspiration of the tracheobronchial tree following the application of saline and a vasoconstrictor into the trachea and bronchi.

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IN MEMORIAM

PHILIP D. KERRISON, M.D.,

1872-1944.

Dr. Philip D. Kerrison, prominent otolaryngologist of New York City, died from a heart attack Jan. 25, at the age of 72 years.

Born in Charleston, S. C., he graduated from State Medical College of South Carolina in 1896, received an M.D. degree from New York University in 1898 and lectured there on aural diseases from 1902 to 1908. From then until 1914 he was Professor of Otology at New York Polyclinic Medical School, and for many years was consulting aural surgeon to Manhattan Eye, Ear and Throat Hospital and Willard Parker Hospital.

He was a Senior Fellow of the American Otological Society, Inc., and a member of the American Academy of Ophthalmology and Otolaryngology.

In 1899, he married Miss Mabel G. Howe, who died some years ago. His daughter, Mrs. Alfred D. Booth, of Scarsdale, N. Y., survives. T. E. W.

LEE COHEN, M.D.,

1874-1944.

Dr. Lee Cohen, of Baltimore, one of the pioneers in plastic surgery of the nose, died Dec. 31 at Johns Hopkins Hospital after a long illness. He was 69 years old.

He was born in Halifax, N. C., graduated from the University of North Carolina and received his M.D. degree from the University of Maryland School of Medicine in 1895. Early in his medical career he became interested in plastic surgery of the nose. He designed several instruments for use in this work and wrote numerous articles on his experiences in this specialty.

He was attending surgeon at Sinai and the Baltimore, Eye, Ear and Throat Hospitals.

During World War I, Dr. Cohen held the rank of Captain, and was in charge of oral plastic surgery at General Hospital No. 11, Camp May, N. J.

In 1914 he became a member of the American Laryngological, Rhinological and Otological Society, Inc., and until recent years took an active interest in its meetings.

Dr. Cohen is survived by his widow and three daughters.

T. E. W.

BOOK REVIEWS.

The Inner Ear, including Otoneurology, Otosurgery and Problems in Modern Warfare. By Joseph Fischer, M.D., Staff Member Beth Israel Hospital, Boston; formerly Associate of Adam Politzer and Senior Otolaryngologist with Gustav Alexander, Policlinic of Vienna, and Louis E. Wolfson, M.D., Instructor in Ear, Nose and Throat, Tufts Medical School; Senior Surgeon in Ear, Nose and Throat, Boston Dispensary. Four hundred twenty-one pages and index, 77 illustrations and seven tables. New York: Grune and Stratton, 443 Fourth Avenue.

This book is a concise and complete account of the anatomy, physiology and pathology of the labyrinth. It is clearly written and profusely and well illustrated.

The authors give the reader the benefit of their own considered opinions and experiences and yet have a complete bibliography.

This book deserves a place in any course for postgraduate students in otolaryngology and would prove a real help as a reference book for practicing otologists. T. E. W.

Acute Infections of the Mediastinum. By Harold Neuhof, M.D., D.S., F.A.C.S., Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons, Surgeon to Mt. Sinai Hospital, and Edward J. Jemerin, M.D., F.A.C.S., Associate Attending Surgeon, Sydenham Hospital, Adjunct Surgeon, Beth David Hospital, Senior Clinical Assistant in Surgery and Research Assistant, Mt. Sinai Hospital. Four hundred and seven pages with index and 157 illustrations. Baltimore: William and Wilkins Co., 1943. Price $6.00.

The authors have treated the subject of mediastinal infections in a novel manner. They not only discuss acute infections of the mediastinum and review the literature but also illustrate the points of their discussions with 100 carefully analyzed cases.

The book is well illustrated with both Roentgenograms and explanatory diagrams.

To anyone whose practice includes endoscopy, this book should prove of real value. T. E. W.

Clinical Audiometry. By C. C. Bunch, M.A., Ph D., Formerly Associate Professor of Otology, Medical School, University of Iowa; Associate in Research Otology, Johns Hopkins University; Professor of Applied Physics of Otology, School of Medicine, Washington University;

Associate Director of Central Institute for the Deaf, St. Louis; Research Professor in Education of the Deaf, School of Speech, Northwestern University. One hundred eighty-six pages with Index and 74 illustrations. St. Louis: C. V. Mosby Company, 3525 West Pine Street, 1943. Price $4.00.

The author of this textbook was well qualified to write it, for there are few men in the country who have as much experience in clinical audiometry as Dr. Bunch, and his experiences are set forth in these pages.

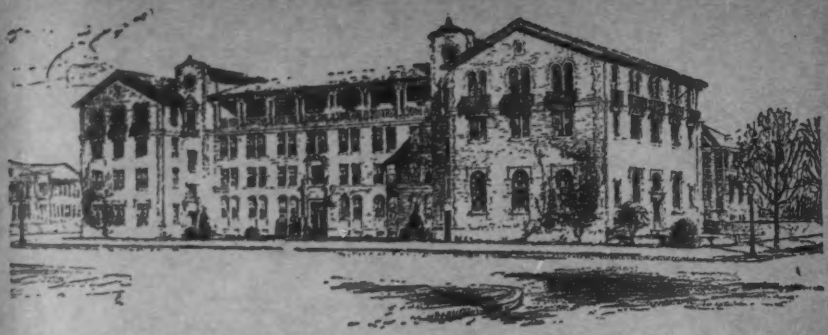
The chapter on "Technique of Audiometric Tests" is worth the time of all otologists, and the chapter dealing with the use of the audiometer in the selection of hearing aids is one which should be read carefully by anyone giving advice to the hard-of-hearing.

The book is short, is exceedingly interesting and well prepared. It deserves a place in the library of all practicing otologists and in the libraries of all medical schools. T. E. W.

Rehabilitation of the War Injured. A Symposium edited by William Brown Doherty, M.D., and Dagobert D. Runes, Ph.D. Pp. 684. New York: Philosophical Library, Incorporated. 1943.

This book is a compilation of articles by numerous individual authors. The subjects considered include Neurology and Psychiatry, Reconstructive and Plastic Surgery, Orthopedics, Physiotherapy, Occupational Therapy and Vocational Guidance and Legal Aspects of Rehabilitation.

The articles are interesting and having them collected together in one book makes them readily accessible for everyone interested in this type of work. The fact that they are all written by different authors is an advantage in presenting each phase of the work in an authoritative manner, at only slight cost in the general cohesion and organization of the book. Its appearance is timely and it is recommended as a convenient and good source of information on the subjects included. F. McD.



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